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*THE JOURNAL OF PERIODONTOLOGY* is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society.

"Truth is what happens to an idea or attaches to it in the process of continual correction and verification."—Louis K. Anspacher.

" . . . The human periodontium includes the gingiva which, in turn, may be described as being divided into two portions: (1) the *attached* gingiva and (2) the *unattached* or *free* gingiva. The latter is frequently designated the 'free gingival margin.' The *unattached* gingiva is a *normal* anatomic finding for man and its presence creates the gingival sulcus. The issue under consideration rests largely on the question of the gingival sulcus and its *normal* depth. The latter *has not* and *possibly never will be* determined as a fixed figure to cover all gingival sulci, in all mouths, at all ages, around all teeth, and in all regions about a single tooth. Anyone familiar with *normal* periodontal anatomy knows that the *unattached* gingiva and the gingival sulcus vary in their dimensions with age and the status of tooth eruption. These dimensions also vary in the different segments of a tooth's circumference, being much deeper on the proximal aspects, less so on the lingual and palatal aspects, and still less so on the buccal and labial aspects. These observations may be made only in terms of a generality and not in terms of exact measurement, since the *normal* is a *range* of *unestablished* limits. The latter point is most important. No one has yet set such limits on *scientific* considerations. . . ."

—Extract from "In Our Opinion" Discussion by Harry Lyons—*See* p. 28.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-EIGHT

# The Clinical Basis for Bone Healing*/**

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THE purpose of this discussion is to bring to bear, where possible, the clinical background in trying to understand bone healing. There are many peculiarities of structure and function which have practical bearing on the problems of fracture treatment and repair of bone.

## THE BLOOD SUPPLY

Evidence that local circulatory supply is important is shown by the peculiarities of behavior of fractures which are in contiguous regions but which behave differently depending upon the adequacy of the local arterial supply. To cite a few instances, let us consider the femoral neck and intertrochanteric region. Intracapsular fractures heal slowly and with difficulty. With pinning, the healing period is a minimum of six months. Even when healed, because of the delicate balance of the circulatory supply, there may be a late necrosis of the head. This may come on two and one-half years after the fracture has healed. Intertrochanteric fractures, on the other hand, can hardly be kept from healing. These heal within two months and scarcely ever affect the nutrition of the femoral head.

Consider again, Colles' fracture of the wrist and scaphoid fracture of the carpus only an inch away. The Colles' fractures heal invariably, the fractures of the scaphoid are a constant source of difficulty for the surgeon; and non- or delayed union is common. The fractures of the anatomical neck of the humerus as compared with the fractures of the surgical neck are peculiar in this sense. In the anatomical neck fractures, all circulation is destroyed. In surgical neck fractures, less than an inch away in the same bone, union with good function results with proper treatment. Where the circulation has been destroyed, the head fragment virtually becomes a sequestrum and unless removed, can lay the basis for a degenerative arthritis.

There are other parts of the skeletal system which may be cited, including the jaw. If you have a fracture of the coronoid process, it heals almost invaria-

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**Read before the American Academy of Periodontology at the Annual Meeting, *Boston, Massachusetts,* Aug. 1, 1947. One of three papers in a Symposium on The Healing of Bone After Injury and Infection. The papers of S. Burt Wolbach, M.D., and of Joseph C. Aub, M.D., both of *Boston,* were not received for publication. Symposium was discussed by Harry Sicher, M.D., *Chicago.*



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bly. If the fracture is in or near the capsule, you may be headed for trouble. You may better anticipate it and excise the temporomandibular joint.

These are instances where relatively gross disturbance of the circulation has taken place. However, it is possible by X-ray and microscopic study to carry these observations further. In fractures of the tibia, it is common to see by X-ray, callus deposition taking place in one region and being absent in another. Usually this disparity can be accounted for by the adequacy of the local circulation. In lower third fractures, the part most likely to show healing is present posteriorly or postero-laterally where the soft part blood supply is most likely to be re-established. This has been taken advantage of for many years in the case of bone grafts for non-union. Clinically, we know that the grafts are much more likely to take if they are placed laterally or postero-laterally where the muscle beds form a source of revascularization.

Microscopically, in experimental fracture healing, an infinite variety of discrepancies in bone formation may be seen. These microscopic variabilities have been described by Sicher (1), Urist (2), and a host of other authors too numerous to cite here.

Whether you explain bone deposition by a cellular, biochemical, or vitalistic concept, the fact remains that somewhere in there, blood vessels are a factor and without microscopic blood supply, bone does not form. Because of the conflicting mass of evidence which has accumulated now for decades, and which has been so ably set down by Sicher and his colleagues, I have begun to seek in still another direction—in the differentiation of the protein matrix. This work, however, is still far from developed and cannot be reported upon here. A few observations, however, may interest you.

### THE NON-CELLULAR MATRIX

Some years ago while working on the adsorption and protection of virus bodies on cell membranes, it became necessary to digest tissue cultures of varying age in order to free the individual cells from the collagenous matrix in which they grew. In the course of this work, which was done at the Rockefeller Institute, a variation in the digestibility of the collagenous matrix was noted (3). The mystery of the formation of collagenous fibers engaged my interest at that time. The variation of the digestibility of collagen fibers due to age and differentiation and other characteristics have been observed, of course, by numerous workers.

I recall these old observations to you only because they are correlated with clinical experience. In an occasional delayed union of the tibia where the injury to the soft tissues is noted at the time of operation and periosteal stripping is excessive, deposition of bone in the fracture site can be quite slow. Yet, because of the excessive soft tissue injury, there may be callus and fibrous healing across the fracture site taking place. If, after four months or so, the fracture line has not begun to disappear, non-union is probable. Yet in a few cases where the deposition of bone did not take place at six months, by re-operating and drilling or fissuring, union was obtained. This can happen in frank non-unions with "sailor's pants" formation of excessive callus and a pseudarthrosis being present.

My interpretation of these phenomena at present is as follows. Somewhere in the course of healing, a disturbance in timing developed. Suppose at three weeks when the alkaline shift for phosphatase activity can be expected that there was still too much surviving injured tissue, poorly vascularized. The fibrous matrix was there and so were cells. Yet bone deposition did not take place. Four months later the conditions for bone deposition were present, but by that time the fibrous differentiation had progressed too far. By drilling or fissuring a non- or delayed union site, fresh injury and irritation to the bone tissue brought a fresh response. In other words, new granulating tissue was brought in and the cycle of repair was repeated. Primitive fibroblasts, fresh new collagen matrix, new capillaries, and all the phenomena of repair recurred. This time union took place. These are incontrovertible clinical phenomena and that is the reason I bring to your attention the non-cellular ossein matrix which is the strut work upon which the rigid osseous matrix is eventually built.

### THE MINERAL MATRIX

This I shall touch upon very briefly because I am sure both my co-essayists may have somewhat to say about it. We know by X-ray diffraction studies, that the orientation of the ossein matrix determines considerably, the orientation of the apatites

which give the matrix its rigid structure. Recently in our laboratory, Dr. E. M. K. Darby working with the X-ray diffraction Geiger counter technic, has confirmed the orientation of the mineral matrix with the ossein matrix.

It is possible to get in adult beef bone a contour tracing which is distinctive for bone orientated transversely, and another, when orientated longitudinally. When the bone is brought to a fine, 200 mesh powder, there are components of both the transverse and longitudinal tracings present, but the distinct orientation contour is incomplete.

This, again, has clinical meaning because we know that when tissue first heals, the orientation of the collagen fibers is every which way. As condition of stress and function begin to bear, orientation of the fibrils increases until the fibrous tissue has replaced, as far as possible, the original tissue present. This is in soft tissue.

Indeed, it is my private speculation that not only can the differentiation of ossein fibrils and their orientation account for the non-cellular matrix of bone and some of its minute structure, but that the linkages of the amino acids which go to make up the proto fibrils of the ossein are important. They may account by their molecular reaction to tension on the part of the ossein, and compression on the part of the apatite; and their molecular or submolecular interaction may, some day, explain the ability of bone to mineralize and demineralize; to toughen under stimulus as in paratroopers and athletes, and to weaken in the sedentary and the aged.

### THE IMPORTANCE OF REDUCTION AND IMMOBILIZATION

Probably nowhere in the skeletal system is accurate reduction more important than in fractures of the mandible and maxilla. There is necessity for good reduction in the weight-bearing lower extremities to preserve axis, length, and rotation. But for the preservation of function, the balance of forces is not so delicate as in the jaws. Certain elbow, carpal, and tarsal fractures likewise require careful reduction in order to preserve good function.

But nowhere is accurate reduction more important than in maxillo-mandibular injuries. I am speaking now of good dental surgery where the surgeon expects to preserve the teeth with good occlusion and not simply to preserve the jaw for a prosthesis. I have not seen a twenty-year follow-up on jaw fractures but even a six months' follow-up has convinced me that reduction without good occlusion is poor dental surgery and a twenty-year follow-up should prove this contention. It is true that there is a certain latitude in mandibular reductions. But this is in terms of millimeters where grinding and adaptation allows for some correction. But perfect reduction with perfect occlusion is still good dental surgery.

### CLINICAL BACKGROUND FOR BIOCHEMICAL FACTORS

There have been certain biochemical factors which have been stressed by Murray and his associates; for example, hydrogen ion concentration. The early observations of pH change made by myself and Dr. Clay Ray Murray were done with quinhydrone electrodes with faulty shielding of the animals and leads. Extremes of 4.6 and 8.3 observed with this technic using a balanced wheat-stone bridge, were almost certainly faulty due to the crudity of equipment available in those days. However, more recent observation with a more careful technic and proper shielding still reveals a shift of tissue pH following injury. One to three days after soft tissue injury not involving fracture, the hydrogen ion concentration is in the range of 6.9 and 7.1; by ten days, the shift is toward the alkaline side, and observations of 7.6 and 7.9 have been made. This again is in soft tissue injury, not in the presence of fracture. These variations of pH may be greater in the presence of fracture. But such redeterminations with improved technic as may be necessary to prove the point have not as yet been done.

I make these comments because of the importance of hydrogen ion concentration in the effectiveness of alkaline phosphatase. It is considered by most observers that for optimum effect on the natural substrate, a pH range approaching 8.3 is necessary. Needless to say, that the alkaline shift is not attained unless good fixation is present and tissue destruction by motion or infection is absent. These facts would be of academic interest only if it were not that clinical behaviour of fractures hinges on them.

### EARLY REDUCTION AND IMMOBILIZATION

Observation of many fractures in long bones at the operating table during open reduction convinces me that "callus" is not necessary for proper healing. I have seen many cases early enough to realize that early reduction with control of hemorrhages and secondary injury may eliminate "laudable callus" entirely. Indeed, if the reduction is anatomic and hemostasis complete, there is no callus whatever. If the postoperative X-rays show that the fracture line is invisible because of accurate reduction, you may be sure that little or no callus will develop(4). The postoperative demineralization by X-ray blends imperceptibly with the vascularization of healing and it is only on the basis of experience in other cases that we know when weight bearing or full use is advisable without risk of re-fracture. The same holds true for any of the long bones of the body.

I mention these things because it is my belief that the formations of pro-callus and callus are excessive reactions of bone to injury or irritation and represent the overaction of the normal response of bone as a tissue. They are not necessary to bone healing provided reduction is early and accurate.

This is particularly true in compound fractures. We know from clinical experience that compound fractures properly débrided, reduced and fixed early do not go on to infection and form very little callus. It must be equally true that early reduction and immobilization of mandibular fractures cuts down the incidence of osteomyelitis and sequestrum formation. Fortunately, unlike many other bones in the body, the maxilla and mandible, except for articular condyles, are in a rich vascular bed. For this reason, it is my belief that if compound fractures of the jaws were seen and treated early, infections could be eliminated. Exception to this is in the cases where the fracture line happens to cross the root bed of a tooth and it is not removed. These, I believe, act as sequestra and form the basis for foci of infection. In other words, without their removal, débridement is not complete. How many times out of ten thousand extractions does one get infection? Yet, an extraction is a compound injury where immobilization is not required.

Phrasing it in another way, because of the vascularity of the maxillo-mandibular beds, development of osteomyelitis following compound fracture presupposes either an error in treatment on the part of the dental surgeon or an accident in time, where the patient was not available soon enough; or, of course, massive destruction of soft tissue with injury to the vascular bed.

In other bones of the body, the surgeon is not so fortunate. In parts other than the jaws, excessive reactions of vascular spasm or vascular injury introduce problems of Sudeck's atrophy, sterile sequestration and delayed or non-union, and a host of other problems which do not plague the periodontist.

### CONCLUDING REMARKS

It is a privilege to appear before this society. I wish to congratulate you as a body to be the first to have sufficient interest in the biology of bone to put on a symposium on bone healing. My remarks have consciously avoided any direct translation of my own clinical and laboratory experience with bone because I believe that the mandible and maxilla are special bones with specialized function and conclusions or generalization on the basis of experience with other bones of the body would not be valid and perhaps not pertinent.

Moreover, I have sought to avoid comment on the pathology or physiology of bone as a tissue because of the far greater competence of my co-essayists, Dr. Burt Wolback and Dr. Joseph Aub.

I thank you, Mr. President, and Members of the Academy of Periodontology for this opportunity to discuss bone healing, and I thank Dr. Frank Beube of the Scientific Committee for inviting me to do so. Like my late Chief, Dr. Clay Ray Murray—when it's about bone—count me in.

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# Concerning the Pathogenesis of Periodontal Disease*

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## INTRODUCTION

THE dental literature is replete with contradictory viewpoints relating to the etiology and pathology of periodontal disease. Investigators have been classed as localists, or those who held that extrinsic factors are all important, and constitutionalists, or those who took the stand that systemic disturbances are wholly responsible. Then again, much argument has centered on the problem of the site of the initial, or so-called primary, lesion. The gingival tissues, the alveolar bone, and the periodontal membrane for many years have had their advocates in this regard. Even within a certain group, while opinions may agree on the site, they may disagree on the source of the etiologic factor**. It would be fruitless, at this time, to discuss these diverse and varied viewpoints at any length†. Many of the early investigators, in attempting to establish a common theory, failed to distinguish between two clinically different and commonly observed types of periodontal disease. Hence, the findings are frequently applicable only to one or the other type.

These types, under various nomenclatures, are so well recognized today that only certain features having a special bearing on our problem will be mentioned at this time. In the first type, gingival inflammation is clinically conspicuous, the pockets tend to develop in a broad and shallow manner and the progress of the disease is slow. Increased mobility of the tooth in its alveolus is relatively a late sign. Essentially, the condition is a progressive gingivitis. This is a Simplex Periodontitis.

In the second type, in its purest form, the gingival tissues show little inflammation clinically. In fact, they may appear at times almost normal, or pale and thin with little tendency towards recession in the earlier stages. The progress of the disease may be rapid. The involved teeth in certain situations are prone to displacement, and diastema may form. The pockets usually tend to develop narrowly and deeply along the root surface, but at times may show an early tendency to widen and encircle the root. Tooth mobility frequently is an early sign. This is a Complex Periodontitis.

It should be mentioned that the dentist is often confronted with cases which are not so sharply defined as these basic types. It is obvious that cases are to be seen which represent admixtures and variations of the fundamental processes concerned. In the second type, for example, the presence of calculus, both subgingival and supragingival, tends to intensify the gingival inflammation, thus helping to obscure the primary process. Under certain conditions, the phenom-

*Read by Dr. C.H.M. Williams before the Amercian Academy of Periodontology, Aug. 2, 1947, *Boston, Massachusetts.*

**For example, Witzel of Germany believed that the initial lesion occurred in the bone crest and regarded the process as entirely local and not dependent on disturbed systemic conditions. Roy of France also concluded that the bone lesion was primary but, on the other hand, considered that the process was brought about by constitutional factors. Local causes were regarded as of secondary importance.

†For a comprehensive review of the subject see Bunting and Hill *(Oral Pathology)*, Lea & Febiger, 1940).

ena of recession and cleft formation in the gingival tissues enter the clinical picture. It is now apparent that the importance of Stillman's Clefts*, especially in their relation to the deeper structures, has not been fully appreciated in periodontal pathology.

### THE ROLE OF INFLAMMATION IN PERIODONTAL DISEASE

As would be expected, the role of inflammation in periodontal disease from the viewpoint of the microscope has been the center of much discussion in recent years. A great deal of the argument regarding the site of a common primary lesion has involved the relationship of gingival inflammation to the underlying bone, as variously observed by different investigators.

The expressed views of a number of workers have not been in agreement in regard to the course of invasion of the supporting tissues of the teeth by inflammatory processes originating in the gingival tissues. However, the accumulated evidence on this problem points to several features of great significance. The first is the relative freedom of the periodontal membrane from inflammatory reaction, as marked by cellular infiltration of gingival origin (Fleischmann and Gottlieb, 1921; James and Counsell, 1927; Box, 1928; and Weinmann, 1941). The second is the tendency of the inflammatory cellular infiltration to involve the bone marrow (James and Counsell, 1927; Box, 1928; and Weinmann, 1941).

Recently, Weinmann (1941) reviewed the subject of the progress of gingival inflammation, and following an extensive histological investigation presented a summary of his findings. These are in substantial agreement with those of the English workers James and Counsell, who reported their findings in 1927. The inflammatory infiltration follows the course of the blood vessels through the gum corium to the crest and periosteal side of the bone. With resorption of the bone, it progresses into the bone marrow. The periodontal membrane is seldom involved by inflammatory cellular infiltration, and when this does occur, the process is one of extension from the bone marrow (Fig 1).

It is of interest to note that James and Counsell concluded that their observations on the pathways of extension of cellular infiltration did little to uphold the concept that lymphatics from the gingival tissues enter the periodontal membrane. It has been commonly accepted that cellular infiltration marks the course of the lymphatics when these vessels are transporting toxic substances such as bacterial toxins.

One manifest conclusion from the foregoing evidence is that inflammation of the periodontal membrane, as marked by cellular infiltration, cannot be held responsible for early and rapid loosening of the tooth in periodontal disease.

It should not be forgotten that inflammation is a defensive process initiated in the tissues by injury, and, as Adami (1910) has

Fig. 1. Pathway of extension of inflammatory cellular infiltration from the gingival tissue over the alveolar crest. Note absence of this small cell infiltration in the periodontal membrane. (Biopsy material.) a. Inflammatory cellular infiltration in gingival tissue; b. cellular infiltration extending over periosteal side of alveolar crest; c. shrinkage artefact; d. alveolar crest; e. periodontal membrane; f. cementum.

*Stillman's Clefts are fissures which occur in the gingival tissues. They extend inwards from the gingival margin along the pocket wall, and outwards to involve the gingival surface for varying distances. The surface defect may appear as a very fine groove which may be fairly straight or curved, and may terminate, fork-like, in two branches.

pointed out, "all means are employed to antagonize the irritant and to effect healing". It is known that enzyme-inhibiting substances are present in the serum of inflammatory exudates (Opie, 1905; Wells, 1920). Thus, it may well be that one of the first defenses against enzyme penetration of the gingival tissues is operative in the pocket when inflammatory exudate is present.

### THE ROLE OF A NECROTIZING PROCESS IN PERIODONTAL DISEASE

Lymphatics are known to exist in the gingival tissues. Their presence in the periodontal membrane was first demonstrated by Schweitzer (1909). Noyes and Dewey (1918) confirmed this finding. According to these investigators, the lymphatic vessels from the connective tissue papillae of the labial or buccal, and lingual surfaces of the gingivae pass over the periosteal side of the bone. On the other hand, the lymphatics arising under the epithelium of the gingival sulcus pass inwards near the cementum surface and penetrate the alveolar crest group of fibers to enter the periodontal membrane. They accompany the blood vessels and nerves and extend in the indefinite connective tissue of Black.

Lymphatic capillaries are formed by thin, delicate endothelial cells and are readily observable in our ordinary microscopic preparations of the gingivae and periodontal membrane.

They are usually demonstrated by special staining methods, e.g., the injection of silver nitrate, or the Gerota mass. With the understanding that in the Dunlop Method of Insufflation the lymphatics are used for the carriage of oxygen to the tissues, we decided to use this method in an attempt to demonstrate lymphatics in the periodontal tissues of sheep. Various substances were employed by filling the sulcus and then insufflating with oxygen. When the dye, toluidin blue, was used, and under the special circumstance of air gaining access to the microscopic section under the coverslip, the lymphatic vessels were clearly demonstrated.

It is significant that, according to certain authorities (Maximow and Bloom, 1930; Drinker and Field, 1933), no lymphatic vessels have been found in the bone marrow.

### NECROTIC TRACTS IN PERIODONTAL LESIONS

In certain cases of periodontal disease, the microscopic sections show fairly long tracts of disorganized tissue in the periodontal membrane. In the early stages, these tracts are homogeneous, and in their course may vary in width and sharpness of outline (Fig. 2). They may pursue a meandering course in the periodontal membrane (Fig. 3) and may approach the surface of the cementum and lie in close proximity to it for a considerable distance apically. The bone surface also may be involved, and the alveolar crest appears to be a region of predilection.

An example is shown in the next illustration where one of these tracts lies close to the cementum surface and adjacent to a nerve fiber and blood vessel (Fig. 4). It is known, of course, that lymphatics usually accompany the blood vessels and nerves.

We interpret these tracts as lymphatic vessels which appear to be distended with a curious coagulum-like material. Another reason, besides their propinquity to vascular structures, for assuming that these vessels are lymphatics, is the occasional presence of typical endothelial cells which follow the axis of the vessel-like tracts.

The loss of the endothelial lining and apparent damage of the adjoining connective tissue seems to be due to the action of a necrotizing agent transported by the lymphatic vessels. In this manner, the course of the involved lymphatics was readily delineated.

Evidence that these tracts are toxic is shown in the next illustration (Fig. 5) by their obvious effect on the cementoblasts. It is demonstrated here that in its course across the periodontal membrane, as the necrotic tract approaches the cementum and extends along its surface, the cementoblasts are lost. This is apparently due to a necrotizing action on these cells. It is to be noted also that there is no evidence of granulocytic reaction in the periodontal membrane, but this is no longer to be regarded as an absence of inflammatory reaction (Menkin).

When the necrotizing process is more extensive and larger regions of connective tissue are involved, the disorganization changes are more marked, as evidenced by the mild vacuolation of the involved tissue. This is

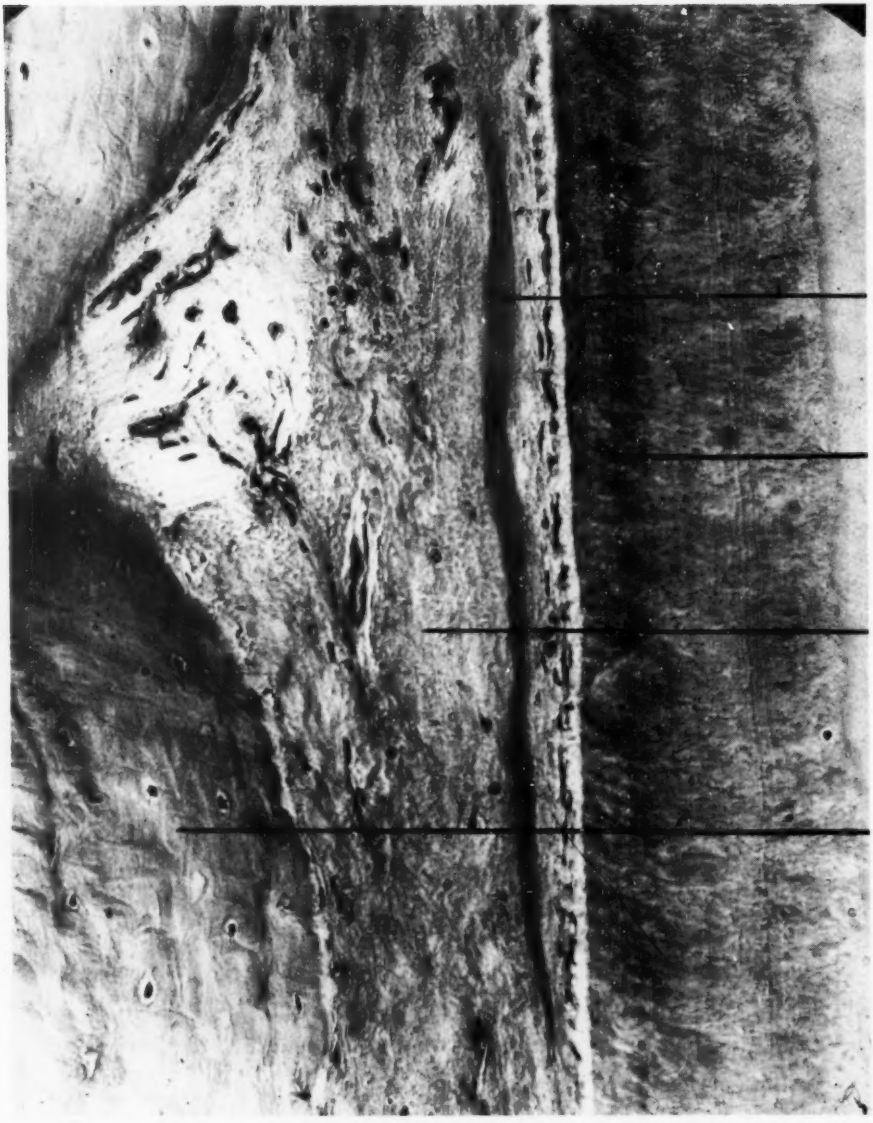


Fig. 2. Long necrotic tract in the periodontal membrane lying fairly close to the cementum surface. The lower end points apically. (Biopsy material.) a. Tract; b. cementum; c. periodontal membrane; d. alveolar bone.

well shown in the next illustration (Fig. 6). It is to be noted, also, that the cementum surface in contact with the necrotic tract is devoid of cementoblasts.

The next illustration (Fig. 7) shows destruction of the alveolar crest through the action of the necrotizing process. With loss of the osteoblasts, and decalcification of the

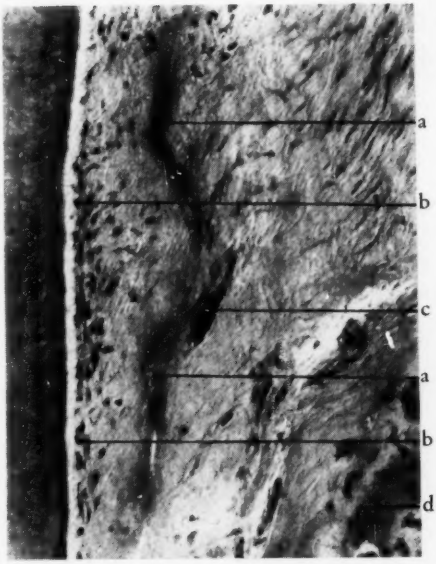


Fig. 3. Long meandering necrotic tract in the periodontal membrane. Note the mild vacuolation in certain regions. The lower extremity of the tract points apically. (Biopsy material.) a. Tract; b. cementoblasts; c. blood vessel; d. alveolar bone.

involved bone, the matrix has undergone vacuolation. Passing from the residue of the bone, a necrotic tract extends across the periodontal membrane to the tooth. It is to be noted that no small round cell infiltration is present.

Necrotic tracts extend from the base of the pocket into the deeper soft tissues. In our next illustration*, at the base of the separated epithelial attachment, the epithelial cells have undergone necrosis. Also, the connective tissue along the tract immediately below the necrotic epithelial attachment is markedly disorganized. Shrinkage artefact accentuates the course of the tract.

### OTHER TRACTS ORIGINATING IN THE GINGIVAL TISSUES

Evidence of tracts having their origin in clefts in the gingival tissues and penetrating deeply into the supporting structures has been observed. The crest of the bone appears to be a site of predilection, and certain examples show the tract extending along the periosteal side of the bone for a considerable distance. Some of the tracts contain squames, epithelial implantation bodies, fungus-like organisms and countless coccoid bodies. There is no associated inflammatory cellular infiltration.

*Not shown in this paper.

### EVIDENCE OF MORE SEVERE AND WIDE-SPREAD LESIONS

We shall now present some of the microscopic evidence of extremely severe lesions as represented by large spaces in the tissues*. These spaces lie in the cemental gingiva and extend laterally over the bone crest, and apically deep into the periodontal membrane. They are of various sizes and shapes and sometimes are alveolate in appearance. It will be observed, that in the region of the "focal lesion", the spaces are almost in contact with the base of the epithelial attachment. At one point, in one example, only one disorganized fiber bundle separates them. The term "focal lesion" is used here to indicate the center of greatest tissue damage. In all probability, it represents the site of the first attack and that from which the disease process has spread.

It will be observed at once that the disease process is not confined to the periodontal membrane, and in fact shows the greatest

Fig. 4. Necrotic tract lying very close to the cementum surface and adjacent to nerve fiber and blood vessel. Note degeneration and loss of cementoblasts. (Biopsy material.) a. Tract; b. cementoid layer; c. nerve fiber; d. blood vessel.

Fig. 5. This illustration shows a necrotic tract extending across the periodontal membrane to the cementum, and apically along the cementum surface. Note the absence of cementoblasts on the involved cementum surface. (Biopsy material.) a. Necrotic tract; b. cementoblasts; c. cementoid layer devoid of cementoblasts; d. alveolar bone.

degree of destruction in the soft tissues overlying the bone. The decreasing severity of the changes as the periodontal membrane is involved points to the unlikelihood of the primary lesion having had its origin in this structure.

As these spaces in the tissues afford little information as to the histopathology basically involved, valuable data in this regard may be obtained only by examining sections of regions at some distance from the "focal lesion". A study of serial sections shows that the disease is a spreading one, and apical and lateral extensions reveal certain details of the early changes.

The larger spaces are the result of the fusion of a number of large vacuoles which were originally filled with a homogeneous coagulum. This was lost in the preparation of the sections. In the more distant sites, the lesions are characterized by hemorrhage and the presence of this coagulum of unknown nature which stains pink with eosin-hematoxylin.

This coagulum tends to be vacuolated, and many red blood cells are to be seen lying on, or near, the borders of the larger vacuoles. Blood vessels are to be observed devoid of their endothelial lining, the red blood cells at times almost filling the vessel lumen. Throughout, the red cells are mainly intact. A conspicuous feature is the absence of inflammatory reaction as evidenced by cellular infiltration (Fig. 8; Fig. 9). Fibrous tissue remnants, in many regions, are still attached to the tooth, and, in one example, in the periodontal membrane, the coagulum fuses with the fibrous tissue, both on the tooth and on the bone. Here, one gains the impression that the blending is the result of fibrous tissue liquefaction.

Another feature to be observed is the disorganization which has occurred in the body of fiber bundles. The fibers show a hyaline-like change with mild vacuolation, which extends for some distance away from the tooth. They appear to be undergoing liquefaction. It is signficant that in the area of attachment to the cementum, no cementoblasts are present. Small vessels minus endothelium and containing red cells are to be noted in the midst of the disorganized fibers, and it seems apparent that a necrotizing agent was liberated in close proximity to these vessels (Fig. 10).

Fig. 6. Marked disorganization of periodontal membrane and alveolar crest. Note vacuolation of involved fibrous tissue in periodontal membrane and also of bone matrix at the crest border. (Biopsy material.) a. Extensive region of disorganization of fibrous tissue in periodontal membrane; b. bone crest apparently decalcified; c. vacuolation of crest border matrix; d. necrotic tract extending along cementum.

## DISCUSSION

It may help to clarify matters at this point by stating our interpretation of these various findings. The mechanism seems to involve the production in the pocket of a necrotizing agent, and under certain conditions, its dissemination to the deeper tissues by way of the lymphatics. When the bone surface is involved, the osteoblasts are killed and bone destruction is carried out. When the cementum surface is contacted, over small or large areas, the cementoblasts are destroyed. With diffusion, or liberation, of the necrotizing agent into fiber bundles, these structures are disintegrated. Hemorrhages are produced through the action on the walls of blood vessels of the necrotizing substance which is liberated into the perivascular tissue. The attacks are probably of short duration.

According to our working hypothesis, the necrotizing agent may contain proteolytic enzymes. Wells (1920), in his studies on the pathogenesis of fat necrosis where extracts of fresh pancreas were injected into animals, found that the first change was a necrosis of surface endothelium. Rich and Duff (1936), in their investigations on the pathogenesis of acute hemorrhage pancreatitis, studied the action of the enzyme

Fig 7 This illustration shows destruction of alveolar crest with marked vacuolation of the organic matrix. Note that a necrotic tract extends from this site across the periodontal membrane to and along the cementum surface. (Biopsy material.) a. Vacuolated matrix of alveolar crest; b. necrotic tract crossing periodontal membrane; c. tract extending along cementoid layer.

Fig. 8. Large destructive lesion involving cemental gingiva and periodontal membrane. (Autopsy material.) a. Cementum surface; b. alveolar crest; c. large alveolate space; d. coagulum showing mild vacuolation; e. blood vessel minus endothelium and almost filled with red blood cells.

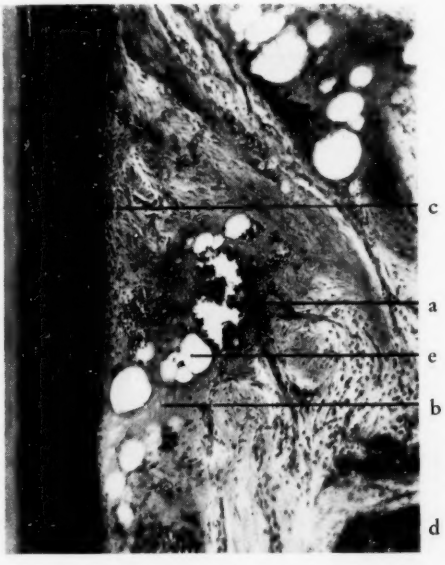


Fig. 9. Large hemorrhagic lesion in the soft tissue above the present alveolar crest. (Autopsy material.) a. Zone of hemorrhage; b. coagulum; c. cementum surface; d. alveolar crest; e. vacuole.

trypsin on blood vessels. They conducted experiments on dogs where solutions of trypsin were injected subcutaneously, and demonstrated that this enzyme has a powerful necrotizing action on the walls of blood vessels.

Menkin (1943, 1946) has described a toxic material liberated by injured cells into exudates, which he has termed "necrosin". This material contains proteolytic enzymes, and it has far-reaching repercussions on visceral organs when introduced into the circulation. It remains to be seen if there is any relationship between our necrotizing agent and Menkin's "necrosin".

As the necrotizing processes, described earlier, damage the attachment apparatus of the teeth, their relation to the clinical picture is marked chiefly by early loosening and displacement of the teeth, and deep pocket formation as a late symptom. In the terminal stages of the disease, the teeth are usually removed without difficulty. One thing seems certain, namely, that this concept of a mechanism involving extrinsic

Fig. 10. Marked disorganization changes in fiber bundle just below base of epithelial attachment. (Autopsy material.) a. Epithelial attachment; b. cementum surface devoid of cementoblasts; c. zone of hyaline-like change and vacuolation; d. small capillary minus endothelium lying in disintegrated fibers.

causes has opened up new pathways in the field of periodontal research.

Time will not permit a discussion of some of the important problems now confronting the research worker. These include studies on the source of the necrotizing agent (Box, 1947); the protective role of inflammation; the repair of the lesions, including the status of pericementitis fibrosa (Box, 1924); and the possible role of anti-enzymatic substances of blood serum and tissue fluids in affecting the action of the incoming enzymes.

## CONCLUSIONS

It is evident that these findings furnish little support of the viewpoint that toxic agents disseminated by way of the blood stream in various systemic conditions are responsible for the damage to the attachment apparatus of the teeth observed in one basic and especially destructive type of periodontal disease.

The immediate mechanism, which to date has been shrouded in obscurity, apparently involves extrinsic rather than intrinsic factors, but this is not to say that systemic conditions, operating as assisting or preventing agents, do not play an important part in the predisposing etiology.

I am under special obligation to Dr. Valy Menkin, Temple University Medical School, Philadelphia, and Dr. M. C. Dinberg, Pathologist, Department of Health, Province of Ontario. Their advice was invaluable, particularly in regard to interpretation of microscopic material. For their many suggestions, interest and courtesy, I wish to express my sincere appreciation.

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# *In Our Opinion*

## IS REATTACHMENT OR POCKET ELIMINATION ESSENTIAL FOR RETAINING PERIODONTALLY AFFECTED TEETH IN HEALTH?

### *Initial Statement*

*By* HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

THE OBJECTIVE of treatment of periodontally affected teeth is to restore these teeth to health and to so maintain them throughout the lifetime. Such treatment has been performed successfully with few exceptions by Riggs and his followers using the conservative treatment. This consists in cleansing and smoothing the detached root surfaces of all deposits and irritating roughnesses, of teaching the patient a home care technic by which the teeth are kept clean and by which the gingival circulation is re-established and stimulated by massage, and of eliminating mechanical traumatic factors by good operative and prosthetic dentistry procedures. Such treatment has resulted in a dense flat gingiva of healthy color and stippling which hugs the tooth tightly. It does not bleed and is not sore when probed and neither shows exudate when pressed nor does deposit which might indicate some seepage, accumulate appreciably on the sulcus root surfaces in six month intervals. The X-ray reveals bone with good cortical plate adjacent to the former pocket area although the level of bone may not be noticeably raised. Little if any recession of the gingiva ordinarily follows this treatment. In most instances reattachment occurs of some type, presumably epithelial, such that the explorer can no longer be thrust the former depth into the pocket. In many cases the pocket is entirely obliterated as far as clinical exploration can determine and a normal sulcus depth only remains. But frequently reattachment is not complete and a deepened sulcus of more or less depth remains even though the tissues in the area are perfectly healthy according to the criteria cited above.

It is admitted that there are cases where conservative treatment is not completely successful and where surgical resection is the treatment of choice. Some cases, mostly in young girls, have such poor recuperative power that the tissues do not return to health even when all phases of conservative treatment seem to have been rendered. Granulomatous areas deep down between the roots of multirooted teeth or between teeth may be so protected by walls of bone or projecting tooth structure that it is impossible to re-establish the circulation through massage by the patient and so they fail to return to health. Also when pockets are very deep and are directed spirally around the root the probability that all deposits will be removed is decreased. Some operators feel that out-of-town patients who can not come back for follow-up treatments at twice yearly intervals can more surely be rid of possible recurrence by resection than by conservative treatment.

The argument is not between those who use only a conservative treatment and those who use only a surgical resection method. Both technics have their

place. It is between those who believe that any deepened sulcus which is demonstrable after a reasonable period following conservative treatment should be eliminated by surgery even though the tissues are otherwise apparently healthy, and those who do not. I am one of those who believe that the fact that reattachment has not occurred when health is otherwise present, is not very important. As long as the patient does his part by his home care technic and will return at twice yearly intervals for check-up and prophylactic treatment to remove any deposits he may have missed or any which may have formed beneath the gingival margins, it is my experience that the tissues will be maintained in health with no signs of further deterioration even though sulci of considerable depth remain demonstrable. I have treated many of these cases over a ten-year period and a few for nearly twenty years. Dr. I. Hirschfeld who has had a longer continuous periodontia practice in one place than I have had, has had the same experience only extended to nearly thirty years. With such a background there is just no room for a pessimistic attitude toward periodontal diseases or their treatment. With the exception of the few cases and conditions where tissue response is negative or the technics of conservative treatment cannot be accomplished, the gingival and periodontal tissues are restored to health and remain so during long periods of observation in the practice and there is no reason to think they will not remain so throughout the lifetime of the patient even though deepened sulci are demonstrable on vigorous probing at each examination. In my experience there is no reason to resort to surgical resection unless it is impossible to restore the tissues to health otherwise. The fact that a demonstrable deep sulcus remains when all other signs indicate health does not constitute such a reason.

There have been a few cases in my practice where periodontal abscesses or acute infective ulcerations have occurred deep in areas where pockets formerly existed or still exist. Almost without exception I have been able to demonstrate in these cases that a mechanical traumatic factor was present and was the reason for the flareup. It had either not been detected or completely treated at the previous examination or had developed since. The fact that such conditions have occurred does not seem to me a valid argument for resection. It is not my experience that the tissues about teeth always remain healthy even after resection if a mechanical traumatic factor is present.

While granting that there are cases and conditions where resection will bring health when conservative treatment will not, it is my opinion that surgical resection is used far more than is necessary or advantageous. There are many disadvantages to surgical resection so that it should not be used when health can be obtained without it. Because surgical resection is interesting and spectacular and can be attempted by anyone with some surgical skill, whereas conservative treatment is tedious and unexciting and a very difficult technic to learn to do well, there has been a wave of enthusiasm for surgical resection as routine treatment for periodontally affected teeth in the United States, which is unfortunate. The younger dentists who wish to become periodontists are often training themselves through the surgical approach alone and consider themselves qualified periodontists when they are completely untrained in the conservative method of treatment. One excuse for choosing surgery on the

part of many who really are well-trained in conservative technic is that deepened sulci often remain after all phases of conservative treatment have been given. In my experience this is not a valid reason.

## *Discussion*

FRANK E. BEUBE, D.D.S.
*New York, N. Y.*

The presence of measurable pockets with healthy gingival tissues, is not an indication of normalcy. There is a constant inflammatory process at the base of these pockets. Large amounts of granulomatous tissue can be shown to be present deep in the pockets, in spite of the fact that the gingiva was firm, stippled, and did not bleed. It is the presence of these abnormal conditions that warrant the elimination of all pockets by some method, be it conservative or surgical. It can be further pointed out that, if periodontal pockets remain after treatment, no new alveolar bone could possibly form in these areas.

The objections to surgical procedures are primarily recession, ugliness, and loss of normal function of the gingival tissues. Mutilation of gingival tissues need not necessarily occur. There are surgical techniques which eradicate pockets and permit esthetics and function of gingiva to remain after healing has taken place. An example of this is Interdental Resection for proximal pockets.

Dr. Leonard states that surgical resection is used "far more than is necessary or advantageous". It is possible that the fault is due to the use of techniques unsuitable for the case to be treated. It is always essential to select a method according to the pathological conditions involved, and not because one is an adherent to the conservative method or to a particular surgical procedure. If any method allows demonstrable pockets to remain after treatment and closure can be obtained by some other technique which will not mutilate the gingival tissue, then it is essential to use that procedure. There are a number of such methods available.

I cannot concur that epithelial reattachment is the end result "in most instances". To the best of my knowledge, experimental evidence supporting this type of repair is lacking. Reppetto reported epithelial reattachment about one tooth after periodontal treatment. This is inadequate for general conclusions. Recent findings by the writer, from experimental and clinical studies, indicate that reattachment which might occur after treatment of periodontosis by both conservative and surgical methods, is of a connective tissue type, with the formation of new cementum, periodontal membrane, and bone.

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C. H. BUCK, D.D.S.
*Dayton, Ohio*

One could qualify the question in many ways and get many answers. If the question may not be modified, my answer is, "No!"

Dr. Leonard has given the question a masterful treatment in his "Initial Statement". I find little with which to disagree, except to say that there are quite a number of records in our files showing mouths restored to health where surgery had previously failed. So, let's not regard it as a last resort.

Diagnosis is of paramount importance. Except in idiopathic cases, the periodontist must have a clear picture in his mind of the etiology, treatment to be selected, and the probable prognosis before treatment is begun. Otherwise, whatever he does will likely fail.

Digital skill in instrumentation is very important and must be acquired by the operator before success in curettage can be expected. Insufficient postoperative care is another common cause of failure. It seems to me that, if thorough work is done within the pocket over a sufficient length of time, other mouth defects corrected, followed by adequate home care, improved nutrition where needed, and regular inspection and correction of small defects as they develop, the depth of the gingival sulcus would be of little consequence.

In our practice, devoted entirely to diagnosis and periodontia, gum resection is chosen only when the time element is of utmost importance.

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EDGAR D. COOLIDGE, D.D.S.
*Chicago, Illinois*

The premise suggested by the subject of Dr. Leonard's "Initial Statement" is not logical. "Is Reattachment or Pocket Elimi-

nation Essential for Retaining Periodontally Affected Teeth in Health?"

Periodontally affected teeth may be those with superficially inflamed gingival tissues, or those having a superficial hyperplasia with a normal alveolar and supporting bone level; while there are other teeth whose periodontal tissue has a deep inflammatory condition with horizontal or vertical bone resorption; or the extreme cases of periodontosis where the alveolar and supporting bone has been lost to an extreme degree.

From the literature that I have reviewed, Riggs, who was quoted as a conservative, was a radical of his time and would probably be classed as a radical conservative even at this time. While he did not use a scalpel in his technique he advocated rough scraping of the pocket tissue with his scalers which caused considerable pain and hemorrhage.

In Dr. Leonard's description of a healed pocket he says, "the level of bone may not be noticeably raised. Little if any recession of the gingiva ordinarily follows this treatment. In most instances reattachment occurs of some type, presumably epithelial, such that the explorer can no longer be thrust the former depth into the pocket".

It is hard to conceive of the existence of a pocket which is cured by any kind of treatment with neither recession of the soft gingival tissue nor a change in the bone level. However, there does occur a shrinkage and tightening of the periodontal tissue against the root surface but not a true organic reattachment of periodontal connective tissue fibers by new cementum. Without organic attachment of the tissues the pocket still remains as a periodontal hazard.

In another paragraph Dr. Leonard says: "I am one of those who believe that the fact that reattachment has not occurred when health is otherwise present, is not very important." It is again difficult to conceive of deep periodontal pockets that rapidly accumulate secretion, exudate, food debris, and microörganisms remaining in health long after having been scaled and polished.

The last two paragraphs express a personal opinion rather than a scientific discussion. If Dr. Leonard is able to get satisfactory results without gingivectomy or any other form of surgical treatment, that is well and good; but, if better and more satisfactory results are obtainable by the use of some form of surgical treatment by another, surely it is not necessary to say that those using that method do so because it is interesting or spectacular. May I quote from Browning who said:

"Now who shall arbitrate?
Ten men love what I hate,
Shun what I follow, slight what I receive;
Ten who in ears and eyes
Match me; we all surmise,
They, this thing, and I, that: whom shall
my soul believe?"

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BERNARD FRIEDMAN, D.D.S.
*Chicago, Illinois*

In my opinion, "reattachment and pocket elimination" are both essential to successful treatment of periodontally affected teeth. However, as both these terms are comparative, it is possible to have shallow pockets remain after treatment if they are innocuous, i.e., if their lining is free of ulceration, hence do not bleed during interproximal massage, and are not deep enough to retain food debris and calcareous deposits.

In the successful treatment of periodontal diseases, no single method alone is adequate.

It is impossible to foresee when beginning treatment if the conservative method alone will be sufficient to restore the peridontal tissues to health, so the successful periodontist may begin his treatment conservatively with thorough scaling, good home care, elimination of traumatic factors, correction of diet and habits, and then find that certain areas have not responded as well as others; usually these are the areas with deep pockets, bifurcation involvement, and areas especially difficult of access. Then, and only then, should surgery be resorted to.

It is only necessary to see one of these pockets opened and to examine its contents to realize that it is fallacious to expect to eliminate all the diseased tissue and crenated bone through an opening as limited as the gingival sulcus.

The area must be laid open for examination and ease of accessability; for if any vestige of the granulomatous tissue in the pocket is allowed to remain or if the alveolar bone is left too crenated to permit reattachment of the gingival tissue the infectious process will return and the treatment will have failed.

The test that the periodontal tissues have been restored to health is the absence of ulcerations in the epithelial lining of the gingival sulcus. This is determined by vig-

orous interdental massage with a tooth-brush and a round hardwood toothpick. In the absence of any hemorrhage it may be assumed that the pockets are innocuous and that reattachment has taken place.

To recapitulate, "reattachment and pocket elimination" are both essential to the successful treatment of periodontal diseases. This is seldom accomplished with any single type of treatment.

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MAX GIESECKE, D.D.S.
*Denver, Colorado*

Having been inculcated with the conservative method of treating periodontal diseases by Dr. Austin James many years ago and having since never been sold on the idea of surgery as a routine procedure, I have no fault to find with Dr. Leonard's "Initial Statement".

I believe that home care should be stressed and impressed on the patient for without it failure is bound to result. It is the operator's responsibility to re-establish circulation but it is the patient who *must* maintain it by regular brushing, massage, and stimulation. After all, it is the function of the blood to nourish, repair, heal, and to combat bacterial invasion.

Where pockets are not too deep to make treatment inadvisable we occasionally use surgery, but more often we use cotton twills in which Zinc Oxide plus subnitrate of bismuth and essential oils, have been incorporated. These are packed in the pocket to distend the tissues to give better access in removing deposits from the roots.

We do not claim to get attachment in most of our cases, but if the tissues are healthy and there is no exudate why resort to electric cautery or surgery? We may have a greater number of attachments than we think but far be it for me to *force* my scalers or probes into former pockets to find out.

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HENRY M. GOLDMAN, D.M.D.
*Boston, Massachusetts*

The objective of periodontal therapy is to restore the affected teeth to health and to so maintain them. The teeth should be functional members of the dental apparatus; the gingivae should be firm, pink, and well attached to the teeth. In order to achieve the above objectives, it is necessary to eliminate all pockets present.

However, before entering into a discussion of this problem, it is best that a few explanations be made:

1. The gingival sulcus may be defined as a space bordered on one side by the surface of the tooth and on the other by the gingiva. The epithelium lining the sulcus is called crevicular epithelium and its attachment to the tooth is called the epithelial attachment. The depth of the gingival sulcus varies; 0 to 6 millimeters readings are found (Orban). Every gingival sulcus may be termed "normal" regardless of its depth if there are no signs of pathology in the investing tissues.

2. A periodontal pocket is a pathologic gingival sulcus, ulceration of the crevicular epithelium and inflammation of the gingival corium having occurred.

3. A periodontal pocket may be classified into two types: (a) a gingival pocket, which may be defined as one in which the base of the pocket is coronal to the alveolar crest, and (b) an intrabony pocket in which the base of the pocket is apical to the alveolar crest.

4. Subgingival curettage is the operation of removing all deposits, cleansing and smoothening the tooth surface, and curetting the soft tissue side of the pocket.

5. Gingivectomy is the operation of surgical removal of the gingival wall of the pocket at a line level to the base.

6. Gingival pockets must be treated in a different manner than are intrabony pockets because of the bone present in the lateral wall of the latter type.

In the treatment of the gingival pocket by subgingival curettage, the objectives are that the ulcerated crevicular epithelium should heal and that the gingival inflammation should subside. In the case of a rather shallow pocket, with resolution of inflammation, shrinkage occurs (since swelling is one of the cardinal signs of inflammation). This results in the elimination of the pocket, a gingival sulcus of very little depth ensuing. However, should the original pocket be of considerable size, then even after shrinkage, the resultant sulcus will still be rather deep. If such a pocket is closed, then it must be done by reattachment of the gingival wall to the tooth surface. This does not usually occur and if it does, most investigators believe it to be an epithelial reattachment. In most cases a deepened sulcus, although not pathologic, (as defined) results. On the other hand, such a

pocket treated by gingivectomy results in a gingival sulcus of practically no depth.

The question Dr. Leonard poses is whether such a deepened gingival sulcus is periodontally harmful to the teeth or whether the tissue should be removed surgically. This cannot be answered without considering other factors such as gingival architecture, position of the teeth in the dental arch, self-cleansing ability of the gingival area, mouth hygiene, and the general health and coöperation of the patient. A deepened gingival sulcus may remain as such in health in one individual whereas in another, because of local environmental factors it soon becomes pathologic. Thus treatment must accomplish not only an elimination of gingival inflammation with resultant pocket elimination, but also a correct gingival architecture. Therefore, it can be readily seen that both subgingival curettage and gingivectomy are accepted procedures to be used by the operator after thorough study of the case at hand and after decision is made as to the tissue changes present.

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HUGH T. HICKS, D.D.S.
*Baltimore, Maryland*

The technique used in the treatment of periodontal disease is a secondary consideration. The primary object is to restore the supporting structures to health and so maintain them.

The result described in the first paragraph of the "Initial Statement" is certainly adequate in every respect. I feel that no surgical treatment is necessary as long as the condition as described exists. The difficult part is to educate the patient so as to have complete coöperation. The most thorough oral hygiene must be practiced at home and regular office treatment received. This is, however, just as true if the case had been treated surgically. Maintain the tissues as described and the periodontium will not be a focus of infection.

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I. HIRSCHFELD, D.D.S.
*New York, N. Y.*

Generally speaking, after periodontal pockets have been successfully treated, the remaining healthy gingival flap forming the pocket may continue healthy indefinitely, even though complete reattachment is not obtained. In other words, the retention of such completely treated tissue is not necessarily conducive to reinfection if oral hygiene and function are maintained to a reasonable degree. This statement is made on basis of periodic examinations of thousands of formerly sick teeth over a period of many years. The exceptions are teeth such as those with trifurcation exposures and others that from the beginning were of questionable or negative prognosis, and would have had to be condemned even if surgical procedure had been employed.

Usually, the depth of the pockets is decreased by a degree of apparent reattachment starting at the bottom of the pocket, or by recession of the gingival margin, or both; but in many instances, the unattached flap hugs the root surfaces so tightly as to actually eliminate a macroscopic pocket. The proof of the successful treatment lies partly in the fact that year in, year out, the gingival tissue forming the pocket remains normal in color, texture, and contour, even about teeth that are mobile and greatly deprived of alveolar support. Often, such post-operative healthy gingival flaps recede rapidly under unduly hard brushing, right down to the point of attachment, but without having ever shown any macroscopic evidence of disease. However, this phenomenon is also frequently produced by hard brushing where there had never existed any periodontal pockets at all, and must, therefore, be considered simply of mechanical etiology.

This should not be understood as indicating that the gingival flaps always recede to a marked degree; indeed, very often they remain so almost completely intact, as to defy detection of a pre-existing infected pocket.

Thus the arguments that pockets must be eliminated surgically or otherwise—does not seem to stand up in the light of ample clinical evidence. Furthermore, the statement often made by some exclusively radical operators that the degree of posttreatment root exposure is about the same regardless of whether or not a gingivectomy has been done, is a statement that should not be made without ample qualification.

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OLIN KIRKLAND, D.D.S.
*Montgomery, Alabama*

The leader of this Discussion in considering the value of reattachment and pocket elimination in the management of periodontal disease has presented a subject of significant importance, not only to the cause of

periodontology, but also in the interest of better general and oral health.

My interpretation of the leader's objective in submitting this subject for discussion is to bring out the merits of conservative subgingival curettage versus surgical resection of the gingivae in the treatment of periodontal disease.

Ability to treat this disease in such a manner as to completely eradicate its cause, regardless of the method employed in the treatment, is a splendid achievement and should be the paramount objective of every periodontist. In the selection of a technique, however, we should remember that there is a right way and a wrong way, an easy way and a difficult way to do all things, and the treatment of periodontal disease is no exception to the rule. It does not matter in the least what kind of technique is used provided the end results are satisfactory to both the patient and the operator. Both parties are chiefly interested in obtaining permanent results regardless of the method used. It is obvious that it would be advantageous to choose a technique that offers the best potentialities. Mistaken judgment in the choice of technique, however, is sometimes equivalent to the difference between success and failure. It would be a forward step in periodontology if a unified method of procedure could be agreed upon and used routinely in both teaching and practice.

I do not agree with our leader in the opinion expressed regarding the innoxious potentialities of "pockets where reattachment is not complete and where pockets of more or less depth remain". It is obvious that such lesions provide an open receptacle for the retention of foreign substances in which bacteria may breed, and in view of the fact that these pockets cannot be thoroughly cleansed each day, it is my opinion that the treatment should be regarded as unsuccessful. Having performed many thousand operations by the surgical resection technique and having observed the postoperative results in most of them, I am happy to say—not without fear of contradiction—but in the interest of better periodontia, that a thorough surgical resection technique would provide for the successful eradication of such pathologic lesions as we have under discussion.

In the second paragraph of the "Initial Statement", our leader says: "It is admitted that there are cases where conservative treatment is not completely successful and where surgical resection is the treatment of choice." If surgical resection is better in certain selected cases we might assume that it could be used to advantage as a routine procedure in periodontia.

He states further: "When pockets are very deep and are directed spirally around the root the probability that all deposits will be removed is decreased, and where patients cannot return twice yearly for follow-up treatments, can more surely be rid of possible recurrence by resection than by conservative treatment." It is my opinion that in all cases where a portion of the supporting structure around the teeth has been resorbed, whether the loss of support has been extensive or whether the lesions have progressed only to a slight extent, surgical resection has for a long number of years, under my observation, proved to be by far the most dependable method of procedure. The extent of the lesion will serve as a guide to indicate the depth of the incision. The overlying gingivae should be removed to a point at or near the bottom of the pocket. When treated in this manner, there is no pocket remaining to worry about.

In the last paragraph of the "Initial Statement", Dr. Leonard says that, "While granting that there are cases and conditions where resection will bring health when conservative treatment will not, it is my opinion that surgical resection is used more than is necessary or advantageous". To use any method recklessly and indiscriminately without regard to diagnosis or prognosis would be evidence of poor training, and those who make such mistakes are not well-informed periodontists. Because a particular technique which has proved itself to be indispensable has been abused by some who are not well-informed in its use, should not reflect upon the technique itself. He further states: "There are many disadvantages to surgical resection so that it should not be used when health can be obtained without it." I do not know what the disadvantages are; consequently, I do not agree with the leader's opinion. My long years of experience and observation, and practice with a surgical technique has led me to the conclusion that there is not the slightest objection to gingivectomy when it is performed by one who is well-versed in periodontology, and has familiarized himself with a well-planned, dependable technique.

ARTHUR W. LUFKIN, D.D.S.
*Hollywood, California*

In response to a request for an opinion on the question, "Is Reattachment or Pocket Elimination Essential for Retaining Periodontally Affected Teeth in Health", I shall first state that my opinion is based on clinical experience, without recourse to histologic evidence, either animal or human.

It seems probable that there will be difficulty in finding a common meeting ground for discussion unless there is agreement on what constitutes health. As an example, a discusser might hold that the presence of a pocket is in itself evidence of disease. If this is accepted as a premise, then it is obvious that further debate must center around methods of eliminating the pocket.

Inasmuch as the so-called conservative method is in many cases the method of choice in my practice, and pockets do exist after treatment, I must then, to be consistent, believe that reattachment or pocket elimination is not always necessary for retaining teeth in health. Without attempting to discuss the indications for surgical procedures I shall also say that they constitute an important part of my practice, and when carefully planned and executed, yield good results.

I am convinced that there is no royal road to success in the practice of periodontia, and that surgical resection alone can be compared to the removal of carious tooth structure without inserting a filling or to placing a partial denture with clasps on carious teeth.

If space permits, I should like to present the opinions of a keenly observant general practitioner, as stated in an editorial in the November, 1947, issue of the *Journal of the Southern California State Dental Assn.*: "There is a deplorable tendency to adopt one-phase therapies for periodontal disease. It is as ridiculous to rely almost entirely upon scaling and polishing as upon removal of traumatizing intercuspation and defective restorations. Advocates of the physiological approach through nutrition and endocrinology (spiced with psychoanalysis) are apt to overlook obvious local factors. But at least these groups are attempting to eliminate the causative factors. Obliterating pockets by the removal of the external tissue wall whether it be by surgery, cautery, coagulation, or caustics, without correcting the causative factors is as futile as shaving to stop the growth of whiskers. As one phase of a multiphase therapy, gingivectomy is invaluable. Sold to a patient as the Alpha and Omega of periodontal treatment, particularly by a 'specialist' it is a fraud that injures our whole profession."

The writer of the foregoing is speaking of an "oral surgeon", who performed a gingivectomy, dismissed the patient after the pack was removed, and made no effort to remove deposits. "There was no evidence (in this case) of any attempt on his part to locate the causative factors, to refer her to any dentist who would search for them, to send her to a dental hygienist for prophylaxis, or even to instruct her in home care. Such incidents constitute a libel upon the good name of Dr. Ward and are the malpractice of oral surgery."

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HARRY LYONS, D.D.S.
*Richmond, Virginia*

Dr. Leonard's "Initial Statement" on this subject is most timely and pertinent. The issue which he discusses has been befogged by invalid contentions much too long and the time for clarification is long past due. Clarification of the question under discussion must rest on certain recognized facts. These involve mainly an anatomic study of the tissues affected by periodontal disease. These tissues collectively constitute the periodontium.

First, the human periodontium includes the gingiva which, in turn, may be described as being divided into two portions: (1) the *attached* gingiva and (2) the *unattached* or *free* gingiva. The latter is frequently designated the "free gingival margin". The *unattached* gingiva is a *normal* anatomic finding for man and its presence creates the gingival sulcus. The issue under consideration rests largely on the question of the gingival sulcus and its *normal* depth. The latter *has not* and *possibly never will be* determined as a fixed figure to cover all gingival sulci, in all mouths, at all ages, around all teeth, and in all regions about a single tooth. Anyone familiar with *normal* periodontal anatomy knows that the *unattached* gingiva and the gingival sulcus vary in their dimensions with age and the status of tooth eruption. These dimensions also vary in the different segments of a tooth's circumference, being much deeper on the proximal aspects, less so on the lingual and palatal aspects, and

still less so on the buccal and labial aspects. These observations may be made only in terms of a generality and not in terms of exact measurement, since the *normal* is a *range* of *unestablished* limits. The latter point is most important. No one has yet set such limits on *scientific* considerations.

One of the most ludicrous contentions offered in this connection is that the normal gingival sulcus should be of "zero depth". Such a contention is obviously self-contradictory and disgusting from a scientific point of view. Yet, it is on this contention that many of those who practice and promote the radical types of surgical periodontal therapy rest their case. It is no compliment to our group intelligence that so many in our ranks have been "taken in" by this invalid contention. It is entirely without scientific support. The histologist who points to a section of tooth and periodontium which may reveal a sulcus of microscopic dimensions forgets that the shrinkage, in tissue fixation, of the soft tissues in such a specimen has much to do with the findings and that these must be viewed in the nature of an artifact.

The question of the dimensions of the normal gingival sulcus naturally leads one to a consideration of a commonly and loosely used term in connection with periodontal disease, namely, "pocket". The originally intended definition of the term "pocket" is a sulcus *pathologically* deepened by periodontal disease and in the tissues of which disease still prevails. Many observers designate certain sulci as "pockets" on the basis of depth alone and without regard for the absence or presence of *actual* evidence of periodontal disease, such as the symptoms of inflammation, etc. This loose use of the term "pocket" is most unfortunate and confusing, so much so that its discard from our periodontal vocabulary appears warranted. On the basis of depth alone, one might well ask the question: When does a normal sulcus become a pocket? The question does not lend itself to an acceptable answer. This indicates how unfortunate is the general use of this term. Even more unfortunate is the contention by some that the selection of radical methods of surgical periodontal treatment is justified by the presence of a "pocket"—a vague structural entity of which a generally acceptable definition or description does not exist.

This discussion serves as a preamble to my agreement with Dr. Leonard that *destructive* and radical surgical methods of periodontal treatment are unwarranted insofar as considerations of the sulcus and its dimensions are concerned. It may be demonstrated easily—by all means of determining states of health or disease—that *good health* can and does prevail in sulci of marked depth. Worded differently, it may be stated that the depth of a sulcus *alone* does not determine the health status of the gingiva or periodontium. Its surgical destruction on this basis is, therefore, unwarranted. That healing and closure of pathologically deepened sulci can be accomplished need not be discussed here. This is a demonstrated fact.

I would go a step further than does Dr. Leonard. He admits: "While granting that there are cases and conditions where resection will bring health when conservative treatment will not . . ." I know of no such "cases and conditions". Much depends, of course, on the judgment and skill of those who use a given method as to what it will accomplish. Ruling out the considerations of marked hyperplastic and frankly neoplastic gingival disease, Dr. Leonard's partly quoted statement is a concession to those who use radical surgical methods for reasons that I personally am not willing to accept as scientifically valid.

Dr. Leonard further admits that ". . . there are cases where conservative treatment is not completely successful and where surgical resection is the treatment of choice". It must be admitted, of course, that no one method of treatment will succeed in every case. However, the failure of one method does not per se make another method the "treatment of choice" for a given case. Obviously, the second method may fail also, as is frequently the case. Much depends on the skill and judgment utilized in the application of either method. Dr. Leonard seems willing to admit that radical surgical methods have superior merit in certain unstated cases. This has not been scientifically established, to my knowledge. For instance, he states: "Some cases, mostly in young girls, have such poor recuperative power that the tissues do not return to health even when all phases of conservative treatment seem to have been rendered." His language here is carefuly couched, but the inference which one may develop is that more radical

methods are indicated or are of proved merit in such cases. If sex and inferred endocrinopathies are factors in these cases, how can one rationalize superior merit for gingival resection over the more conservative subgingival curettage? How can one or the other affect an underlying endocrinopathy and so afford a superior result?

Dr. Leonard also brings in the question of massage by the patient as a factor in determining the selection of treatment methods and results. The question of massage in this field is sorely in need of critical evaluation. A discussion of this problem would extend this commentary beyond reasonable bounds. At this time, it may suffice to state that here is another example of a procedure widely promoted and used, and for which there is much *testimony* but no *scientific evidence*. There is an important difference between the two, not generally appreciated by practitioners at large.

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JOHN OPPIE MCCALL, D.D.S.
*New York, N. Y.*

The discussion of the necessity of pocket elimination in periodontic treatment is very timely and valuable. I am in basic agreement with Dr. Leonard's contention that it is permissible to retain teeth on which pockets persist after otherwise successful treatment, always assuming that conditions are suitable for maintaining health. By that I mean essentially what Dr. Leonard says in his statement, i.e., that the tissue is healthy, the tooth firm and readily kept clean on its exposed surfaces, and furthermore that the patient is in a good state of health.

I agree with the statement that "there is no reason to resort to surgical resection unless it is impossible to restore the tissues to health otherwise". The experienced periodontist can usually determine in advance those cases which are likely to require resection and plan his treatment accordingly. Even when he finds he has not secured the favorable result he anticipated with conservative treatment he can still fall back on surgical resection. The opposite procedure is, of course, not possible.

Surgical resection has inherent disadvantages not found in the conservative method, hence I feel it is always in the patient's interest to use the conservative method first unless conditions are such that it is obvious at the outset that it cannot succeed. I object particularly to surgical resection on the upper anterior teeth because of the unsightly appearance produced and I question the long range acceptability of acrylic facings to cover root surfaces exposed by that procedure as recently advocated by Orban (Orban, B., Periodontal Disturbances: Practical Management, *J.A.D.A.* 35(8): 545-556. Oct. 15, 1947). Also these teeth, of all in the mouth, are most likely to respond to suitable conservative treatment and to develop a reattachment. In that regard, I agree with the statement that reattachment of some suitable type is often obtained as a result of conservative treatment. I regret the statements made by Orban (in the same article) to the effect that reattachment is not to be expected in chronic types of periodontal disease which, he says, constitute about 99 per cent of cases with deep pocket formation. This is far from being my experience, both in my earlier years of practice and recently.

I agree that conservative treatment is tedious and requires the development of a special combination of refined tactile sense and mental visualization of the unseen area which is being treated. Without derogatory implication I think it can be said that some excellent dentists will find the development of this technic so laborious and difficult as to make the effort to acquire it impractical. For these dentists surgical resection is far better than unfruitful conservative treatment. The fact remains that for all but a small minority of cases conservative treatment can be conducted successfully according to the standards set up in the statement and, if successful, is by all odds the method of choice.

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JACOBY T. ROTHNER, D.D.S.
*Philadelphia, Pennsylvania*

The pertinent question, "whether reattachment or pocket elimination is essential for retaining periodontally affected teeth in health" depends, in my opinion, on several conditions. One, is the location of the pocket. By this I mean, is the pocket situated in an area that will catch and retain food? Will it lend itself to cleansing by ordinarily accepted means? Two, can and will the patient maintain these areas in a state of health? Three, is the anatomy and position of the affected tooth such that this member of the arch will assist in preventing further ingress of debris during the normal action of mastication? Four, is the patient

subsisting on a soft adherent type of diet or one that is in itself an aid to the cleansing and stimulation of the part?

The statement that "little if any recession of the gingiva ordinarily follows this treatment" I believe should be qualified. This depends upon the type of condition that is present and should take into consideration the height of the remaining bony support. I have seen cases of complete and careful curettage of pockets and institution of stimulation, followed by a marked recession of tissue. One would almost be led to believe that radical surgery had been performed. This condition I admit is not too often seen, but nevertheless cannot be overlooked.

I concur with the opinion, that if the tissue remaining, will closely adapt itself to the tooth, the color and texture being good and appearing to be in all respects normal, there is no necessity for its removal, leaving an unsightly appearance of the teeth. I too, can back this with clinical cases that have been under observation for ten years or more. The greatest degree of success has been with those pockets that were located on either the facial or lingual aspects of the tooth. Proximal pockets were not quite as successfully handled. In areas where too great a loss of septal tissue has occurred, patients may not always follow instructions completely in trying to keep these areas free of food particles. This results in a progressive degeneration of tissue. Root proximity must also be taken into consideration.

Nothing was mentioned regarding the removal of the pocket lining in the first paragraph of the "Initial Statement". I infer that Dr. Leonard feels that this is unnecessary in the proper cleansing of a pocket. If so, to this I cannot subscribe, as in my experience it is as necessary to curette the tissue side of a pocket as it is to plane a root surface. This is too important to be overlooked.

In all instances, I believe that the conservative type of treatment should first be instituted, and if carried out in the skilled manner in which it should be, there is not so great a need of surgery as some would have us believe.

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SAUL SCHLUGER, D.D.S.
*Jersey City, N. J.*

I am one of those who believe that pocket elimination either by closure or resection is a necessary requirement for successful treatment. This is a general statement and is subject to some rather unusual exceptions as are all general statements. However, for the purposes of this discussion it will serve as a basis for a viewpoint.

My points of contention with the "Initial Statement" are several. In the first place it describes a series of clinical pictures which are in themselves rather uncommon and it sets these up as the average picture. In the very first paragraph "a good cortical plate" on the margin of the affected bone is claimed for the average case. It has not been my experience that this phenomenon occurs even after successful treatment in most cases. The statement is also made that little if any recession occurs after curettage. That also is open to serious question. We are not discussing a single phenomenon here but are dealing with a complicated disease complex of varied etiology and presenting just as varied a clinical picture. In those cases which present heavy subgingival calculus and rather violent gingival symptoms, recession does occur as a general rule. And it is not a minuscule recession either but a definite recognizable one which on occasion is extreme. It is precisely in these cases that curettage serves most successfully to eliminate the pocket.

The most difficult and stubborn cases are those which present a very similar picture *before* treatment to that outlined above as an acceptable end result. I refer to those pockets in which the overlying gingiva is flat and stippled and shows no clinically visible inflammation. There is no subgingival calculus and there may not be suppuration. These cases respond rarely to curettage by closure.

I believe that all these deep gingival sulci should be eliminated either by closure of the pocket or by resection of the unsupported gingiva if curettage should fail. I feel that to allow such a sulcus to remain is to allow a nidus for infection and tissue breakdown to remain. It is not enough to establish a clinical appearance of health in tissues. To me it becomes a question of the degree of chronicity that is established. The fact that the teeth may be retained for a long period under such treatment does not alter the essential point that many other diseases may be reduced to mild chronicity over a long period of time. Since the means exist to eliminate

the sulcus they should be used. I know I am not the only one to attach such importance to pocket elimination. Those who do not agree with the surgical approach are very proud of their cases of reattachment. They do not seem to think it is of little importance as has been stated in the "Initial Statement". If it is really of such minor importance why go to such length to achieve it? The only possible answer is that it *is* important and desirable—and only because sulcus depth is zero.

Most multirooted teeth that show pockets between their roots are not favorable subjects for surgery. Except for some lower molars the prognosis of these teeth is always bad. That brings up the point of surgical failures. Poorly conceived and poorly executed surgery will fail just as will any other technique improperly developed. There is no substitute for diagnosis and judgment. The primary aim of treatment is pocket elimination. I believe that aim should be realized.

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JUSTIN D. TOWNER, D.D.S.,
*Memphis, Tennessee*

The question presented in the "Initial Statement," the essentiality of reattachment or pocket elimination, is one easily answered. When, however, methods of procedure are imposed the discussion becomes more involved. Periodontists are more in agreement that establishment and maintenance of a normal gingival sulcus is requisite to periodontal health than in the choice of a means by which it may be accomplished.

Few deny that a sulcus 3 mm. or more, in depth is either diseased or will become so, since it serves as an incubator and as such constitutes an actual menace to health. For this reason gingival recession which keeps pace with alveolar resorption is a good fortune in that there is no pocket formation to hasten the destructive process.

Those who prefer the removal of chemical and mechanical irritants, including adjustment of occlusal stress, and smoothing denuded root surfaces must depend upon rapid regeneration of epithelium within the sulcus as a barrier to the invasion of bacteria and the diffusion of toxins. Those remaining in the tissue and remnants of degeneration must be disposed of by leukocytes and lymphocytes. Round cells, capillary vessels and fibroblasts aid in the rebuilding program.

The pronouncement of many histologists is that suppurative detachments are permanent detachments and that hope of preserving the adjacent alveolar process, or rebuilding its cortical plate, lies within the integrity of the periodontal membrane. If this be true there is at least no certainty of physiologic reattachment.

The proportion of failures in substantial recovery by means of subgingival curettage alone has led to a more definite method of pocket elimination—that of gingival resection. Periodontists who adopt this surgical practice are conscientious in their effort to effect a postive cure. They are not exhibitionists, to whom the spectacular appeals; they merely make a so-called radical approach to conservative treatment.

Since recovery and substantial resistance to recurrent disease, demands elimination of the nidus for bacterial propagation and accessibility of the area to subsequent hygienic stimulation, a normal sulcus should be established. This, to me, means pocket elimination.

## *Concluding Statement*

I think this discussion has been a valuable one in that it has served to bring out the theoretical bases on which different procedures rest. I want to thank those who have so cordially coöperated by sending in their discussions.

One idea expressed by Beube as a reason for resection in all cases of deepened sulci seems to me faulty. He says "The presence of measurable pockets with healthy gingival tissues is not an indication of normalcy. There is a constant inflammatory process at the base of these pockets. Large amounts of granulomatous tissue can be shown to be present deep in the pockets, in spite of the fact that the gingiva was firm, stippled and did not bleed".

I agree that pink, firm appearing, stippled gingival tissues may be present on the surface with granulomatous masses underneath, but such superficial appearance should not be the criterion of health of the underlying tissue. In the "Initial Statement" additional criteria were: "a dense flat gingiva which hugs the tooth tightly. It does not bleed and is not sore when probed and

neither shows exudate when pressed nor does deposit, which might indicate some seepage, accumulate appreciably on the sulcus root surfaces in six-month intervals. The X-ray reveals bone with good cortical plate adjacent to the former pocket area although the level of bone may not be appreciably raised". Such criteria indicate to me that underlying granulomatous tissue has been healed and replaced with normal healthy connective tissues. Unless I get such evidence I do not consider that a healthy result has been obtained.

In the January 1933 issue of this Journal (pp. 33-47) Dr. Hirschfeld had an article "The Dynamic Relationship between Pathologically Migrating Teeth and Inflammatory Tissue in Periodontal Pockets". In this article he depicted the effects of granulomatous tissue in deep pockets between roots in separating teeth. After conservative treatment consisting of root surface curettage and home care procedures, these separated teeth came together again without any orthodontic interference. Similarly teeth protruded because of granulomatous tissue about lingual pockets have retruded into line when proper conservative treatment was directed to eliminating irritation and reducing congestion by massage. In some cases a depression or groove in the gingiva remains after treatment where underlying granulomatous tissue formerly caused a bulge. Such evidence indicates that efficient conservative treatment causes underlying granulomatous tissue to become healthy without resection or curettage.

I do not agree with Friedman and Rothner that curettage of this granulation tissue is necessary in order to make it healthy. Granulomatous tissue is only disordered connective tissue and is perfectly capable of being restored to health when the foreign body irritants that brought it about have been removed and the circulation has been restored to normal by the stimulation of compression and release massage. The reason why such tissue does not always heal is, with rare exceptions, either because irritating factors are not removed from the tooth or because the granulomatous tissue does not get massage which in turn may be due to inefficient home care or because the granulomatous tissue is too inaccessible to be reached by the compression of the home care technic.

I agree with Lyons that the zero depth objective for gingival sulci is unsound as human beings normally have a free marginal gingiva with a definite gingival sulcus. Some inflammatory cells such as lymphocytes and plasma cells are practically always found on microscopic examination in the tissue about the base of the sulcus. Yet no one advocates the resection of free marginal gingiva in all cases.

I also like Lyons and Goldman's comments in which a sharp differentiation is made between a pocket as being a deepened sulcus with disease about it, and a deepened sulcus about which no appreciable disease is present. In my opinion even after hearing the evidence for resection, the deepened sulcus remaining after successful treatment of the pocket is not a menace to the patient either locally or systemically and its presence should not be considered a reason for resection as a routine procedure.

*—Harold J. Leonard*

# *Editorials*

## Socialized Health Service in Practice

MUCH has been said and written regarding socialized health service, including dentistry—a matter which should be of considerable interest to the dental practitioner.

It is pointed out that more than half of the people of this country receive little or no dental care. All are agreed that this is a regrettable situation, but what should be done to correct it is still a debatable question. There are those who are of the opinion that socialized health service is the answer. To this end legislation in various forms has been sought with a view to providing such service at the expense of the taxpayer. It might be enlightening, therefore, to inquire just how it has actually worked out in those countries where it has been given a fair trial.

Such a study is available in a report from New Zealand, where state health service has been in operation for ten years.* A review of the experiment has recently been made by the New Zealand House of Representatives in which it is reported that it is "common practice for physicians engaged in the health service to undertake treatment of an impossible number of patients and to push them through at the rate of one every five minutes," also that "there is a definite falling off in the standard of medical practice from both the ethical and the clinical points of view." This is attributed to the absence of incentive to keep abreast of professional progress, which "has resulted in a serious dearth of skilled physicians and surgeons, and consequent discouragement of both medical research and postgraduate study." As a further cause for dissatisfaction is the mounting cost of such service. For the year ending March 1, 1947 the cost to the people of New Zealand was $139,000,000.00 as against $83,000,000.00 for the preceding year. Other costs, such as drugs, medical benefits, etc., were correspondingly increased. The Minister of Health when confronted with the situation "admitted that the abuses of the system were such that the government must seriously consider altering it."

It might be argued that this could not happen here, but since human nature is about the same the world over, there is no assurance that it would not.

As long as the people of this country are willing to pay for tobacco two hundred million dollars more each year than they pay for combined dental, medical and surgical care (as is the case at present) is it unreasonable to believe that many more than do, should be able to pay for needed health care?

—A.H.M.

*J.A.M.A. 135:447, Oct. 18, 1947.

# Importance of Periodontology Recognized

AN Editorial in the June 1947, issue of *Dental Abstracts** calls attention to the extensive research in periodontology and closely related subjects that is being conducted in the various dental schools and other institutions in the United States, Canada, and other countries. The December 1946, meeting of the American Association for the Advancement of Science (Section of Dentistry) is recalled, the program for which was made up entirely of a symposium on periodontology. Also mentioned is the progress that has been made by the Dental Educational Council of the A.D.A., in coöperation with some of the Specialty Boards, in their study of ways and means to regulate and to supervise the training and certification of several dental specialists, including periodontists.

That these events have had their influence on dental practice is proved in one instance by the fact that the official organ of a national dental society, made up mostly of general practitioners, devoted its October 1947, issue exclusively to periodontia. The National Dental Association which was organized to "embrace every colored dentist of the nation" held its first meeting in 1933. In the issue of their Bulletin, before mentioned, there were eight well-written articles which discussed the subjects following: Periodontia in General Practice; The Effect of Operative Procedures on Periodontal Conditions; Introducing Periodontia Into Your Practice; Etiology of Periodontal Disease; Partial Denture Construction and Periodontia; Occlusion; Modern Treatment of Periodontal Disease; Instrumentation in the Treatment of Periodontoclasia. Other articles on periodontia are awaiting publication.

This organization should be encouraged in its recognition of the fact that periodontia is fundamental in the correct practice of dentistry. If this truth is stressed in future issues of the Bulletin and in programs for future meetings the continued development of the members of this society in the way of successful service in dentistry is assured. We commend the Editor and his staff, one of whom is the new Dean of the School of Dentistry—Meharry Medical College, Clifton O. Dummett, B.S., D.D.S., M.S.D., M.P.H.

—G.R.S.

*See p. 39 this JOURNAL OF PERIODONTOLOGY

MEMBERS OF A.A.P.—Please note first announcement of the Eighth Postgraduate Seminar pp. 37 and 38—this Journal.—ED.

# Nutrition and Dental Health

*Selected*

*By* DOROTHEA F. RADUSCH, D.D.S., B.A., M.S.

## BICARBONATE AND RENAL REGULATION OF ACID-BASE BALANCE

*Condensed* from *J.A.M.A.* 133:777, Mar. 15, 1947

THE KIDNEY has an important role in regulation of the acid-base balance. The effect is manifested by the reabsorption of base, the excretion of acid phosphate and other acidic anions, the synthesis of ammonia and the formation of citric acid, and the reabsorption of bicarbonate.

Pitts and Lotspeich (*Am. J. Physiol.* 147:138, Sept. 1946) studied the latter role. They followed the quantitative relationship between plasma bicarbonate concentration and the rates of tubular reabsorption and urinary excretion of the bicarbonate. The results indicate a definite "renal threshold" for bicarbonate. Below this practically all the bicarbonate is reabsorbed, whereas above the threshold, bicarbonate is excreted.

This threshold is relatively stable. The quantitative efficiency of renal reabsorption was illustrated by a calculation showing that under ordinary conditions in which there is a need for base conservation in man, 99.95% reabsorption of bicarbonate may occur. In contrast, in case of base excess, bicarbonate excretion may be almost equally complete, as has been observed on certain therapeutic and experimental regimens in which more than 100 Gm. of sodium bicarbonate has been ingested and excreted per day.

The renal reabsorption and excretion of bicarbonate constitutes a primary mechanism whereby the body may defend itself against either loss or expansion of its alkali reserve and thus maintain its acid-base balance.

## THE MOBILITY OF BONE

*Quoted* from *J.A.M.A.* 135:514, Oct. 25, 1947

The introduction of the use of stable isotopes in experimental biology soon resulted in the demonstration that protein of the body exists in equilibrium with the tissue amino acids and that it is constantly being broken down and resynthesized. That this kind of equilibrium exists also between the apparently inert bones and inorganic ions is shown by accumulating evidence. In 1945 it was reported (*J. Biol. Chem.* 159:411) that precipitates which are formed from solutions of the approximate inorganic composition of blood have a composition similar to the inorganic portion of bone and that the solubility of these precipitates corresponds closely to the amount of calcium and phosphate present in the plasma. The exact composition of these inorganic precipitates varies with the composition of the supernatant solutions, in much the same way that the composition of bone varies with the inorganic composition of the plasma. The experimental use of radioactive phosphorus resulted in demonstration that the phosphate of bone is rapidly exchanged in the living animal with the phosphate of serum; this exchange with whole bone occurs almost as rapidly as the exchange between powdered bone and the surrounding solution. Since the reaction

appears to occur on the crystal surfaces of the bone, some mechanism apparently facilitates diffusion of the phosphate through the bone. These experiments show the exchange relations which exist between the bone and the tissue fluids and also support the thesis that the diet influences the healing of fractures and the health of bones.

* * *

## EFFECTS OF MINERAL OIL

A recent editorial in the *Journal of the American Medical Association*, (135:512, Oct. 25, 1947) again warns against regular use of mineral oil either as such or in a mayonnaise or oil salad dressing. It cites evidence of decrease in plasma carotene in human subjects using varying amounts and forms (Alexander, *et al: Proc. Soc. Exper. Biol. & Med.* 65:275, June 1947).

Furthermore, Alvarez (*Gasteroenterology* 9:315, Sept. 1947) in reviewing work on absorbability of mineral oil cites research showing that emulsified forms in common usage are about one-half absorbable. Lesions in the liver and lymph nodes of animals were related to ingestion of mineral oil.

These observations raise the grave question, Alvarez says, whether mineral oil can be used safely, year in and year out, as some persons use it. They also raise the question whether purveyors of food should ever be allowed to substitute mineral oil for the edible fats. The Council of Foods and Nutrition of the A.M.A. concluded in a report (*J.A.M.A.* 123:967, Dec. 11, 1943) that the indiscriminate use of mineral oil in foods and cooking is not in the interest of good nutrition.

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# Announcement

### *Eighth Postgraduate Seminar*

## THE AMERICAN ACADEMY OF PERIODONTOLOGY

April 12, 13, 14, 1948

*Sponsored by*

UNIVERSITY OF MINNESOTA, SCHOOL OF DENTISTRY

To be held at the Center for Continuation Study University of Minnesota

PLAN NOW to attend.

DR. LAVELL HENDERSON, Dept. of Biochemistry, University of Wisconsin, will present a general survey of the importance of proteins in nutrition, the newer knowledge regarding amino acids, the improved methods for measuring amino acids in foods and biological materials, the possible amino acid imbalance, etc.

DR. JANE LEICHSENRING, Dept. of Nutrition, University of Minnesota, will discuss mineral metabolism with a report of research on human subjects.

DR. MAX O. SCHULTZE, Dept. of Biochemistry, University of Minnesota, will present the subject "Some Fundamental Aspects of Iron Metabolism".

DR. C. S. STULBERG, Dept. of Bacteriology, University of Minnesota, will lecture on "The Nature of Viruses and Virus Diseases".

DR. HARRY GARVIN, Winnipeg, Canada, will discuss "Clinical Periodontia".

DR. GRACE ROTH, of the Mayo Clinic, Rochester, Minnesota, who has done most of

the original study at the Mayo Clinic on the effects of smoking, will discuss the experimental findings. This will be valuable because of its factual nature.

There will be several other speakers for whom final arrangements are incomplete upon going to press. Watch for further details in your separate announcement. When you receive registration blanks, please respond promptly, since reservations will be limited. Save the dates now—Monday, Tuesday, Wednesday, April 12, 13, 14, 1948.

Dorothea F. Radusch, *Chairman*
Program Committee for Seminar

## An Ideal for Dentists

Reprinted from the *Jour. Kansas State D.A.* 31 (4): 7. Nov. 1947

Speaking before the Italian Dental Association, Pope Pius XII had the following to say regarding dentists and their profession:

"Dentistry requires an exact acquaintance of, and experience in, the sciences and arts. It demands tact, intuition, and psychological finesse in order to acquire the art of persuasion, and that the moral authority necessary to anticipate and overcome those instinctive fears and hesitations on the part of the patient, more distracting than actual pain. You need much patience, great physical resistance—you have to sustain a perpetual effort of restraint of all your senses, your nerves are strained, your body, your mind, your will and your sensitiveness. Always standing, often in a constrained attitude, your eyes are strained, both hands are busy, they must be supple with the fingers contracted with the manipulation of several instruments at one time, every movement impeded by reflexes and reactions on the part of the patient, which are not always possible to perceive. And all this time you must remain unperturable, calm, courteous, gentle and full of charity."

## Extract from Editorial

"PERIODONTOLOGY" DENTAL ABSTRACTS 3(3): JUNE 1947

Research in periodontology is outstanding in certain institutions because of the presence there of enthusiastic research workers or because a general research program including periodontology is well supported.

The University of California is doing valuable work. Herman Becks, W. W. Wainwright and associates are conducting animal and human experiments with endocrine and nutritional factors as causative agents in periodontoclasia. James Nuckolls and R. W. Rule, Jr., are working on bone physiology as it applies to the periodontium.

In Chicago, Balint Orban is doing brilliant work in histopathology. J. P. Weinmann and Harry Sicher are studying the physiology and pathology of bone, especially in relation to the teeth and jaws.

At Northwestern University, L. S. Fosdick and associates are studying saliva, salivary deposits and plaques—important work in the field of periodontia.

Isaac Schour and Maury Massler at the University of Illinois are including gingival and periodontal disturbances in the range of their excellent research activities. The eruptive process and its relation to the anterior pituitary growth hormone is the basis of one study. Gingivosis, a peculiar gingival disorder observed in malnourished children of Italy, is another.

At Tufts College in Boston, Irving Glickman is studying diabetes in relation to periodontoclasia. He has also made a study of periodontal conditions in the malnourished people in Poland.

At New York University, S. C. Miller, Sidney Sorrin and their associates are studying occlusion and its relation to periodontal health and disease.

Columbia University has several researches in progress. F. E. Beube is working on methods of stimulating bone formation where it has been destroyed around the teeth. Mr. Karshan and B. Tenenbaum are engaged in blood chemistry studies of individuals predisposed to periodontoclasia and Vincent's infection. T. Rosebury is working on the bacteria and antibiotics of significance in gingivitis, periodontoclasia, and Vincent's infection. M. Diamond and E. Applebaum are studying growth processes about the teeth and jaws. L. R. Cahn is studying the pathology of soft tissue lesions and tumors of the mouth. L. A. Cohn is studying occlusion and mouth rehabilitation in periodontoclasia cases and D. E. Ziskin and associates are doing animal experimental work in the field of endocrines and vitamins and their relationships to gingival changes.

Paul A. Boyle is doing excellent work at the University of Pennsylvania in animal experimentation, studying the relation of vitamin deficiencies to periodontal disturbances.

At Baylor University, B. Gottlieb is studying the effect of occlusal disharmonies on the histopathologic picture.

F. Krasnow at the Guggenheim Dental Clinic in New York is doing important work on nutritional relationships to periodontal health and disease. E. G. Hampp at the National Health Institute at Bethesda, Md., has done outstanding work on the fuso spirochaetal flora of the mouth. Henry Goldman at the Massachusetts General Hospital has contributed important findings on histopathology of periodontoclasia.

Outside the United States, important work is in progress at the University of Toronto, Canada, by Harold Box, C. H. Williams, and associates on calculus formation, pathologic effects of traumatic occlusion and other phases. R. G. Agnew and Mary Agnew of West China Medical College have made important contributions relating to nutrition as it affects the teeth and periodontal structures. E. W. Fish of London, England, is working on the infectious aspects of periodontal disease. He has recently published an excellent book, "Paradontal Disease—A Manual of Treatment and an Atlas of Pathology." Another excellent book but one published in Spanish is "El Paradencio" by Francisco M. Pucci of Montevideo. A magazine, "Paradentologie," has just started publication in February. It is published by ARPA Internationale in Geneva, Switzerland, and is printed in French. Work which was in progress before the War in Berlin, Vienna and other European centers has either ceased or has not been made available here. China, India, Russia, Australia, New Zealand and other countries not specially mentioned, with the possible exception of Sweden and Norway, are on the whole not as far advanced in any branch of dentistry as the United States and have not recently made any significant contribution in the field of periodontology. —H.J.L.

# News Release

COLUMBUS, O., Jan. 1948—Greater development of teaching in the field of oral cancer at Ohio State University's College of Dentistry has been made possible through a grant of $4,990 from the U. S. Public Health Service.

Announced by Dean Wendell D. Postle, the grant is one of the first to a dental college by the Research Grants Division of the National Institute of Health. Administration of the expanded cancer teaching program will be handled by Dr. Hamilton B. G. Robinson, professor of oral diagnosis and pathology, OSU College of Dentistry.

The grant, recommended by the National Advisory Cancer Council, will facilitate the teaching of pathology, diagnosis and therapy of oral cancer by making possible the expanded activity of present personnel and employment of new personnel.

"Clinics, microscopic conferences, seminars and lectures will be used", Dean Postle said, "these will emphasize the importance of cancer, in general, and oral cancer, in particular."

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## *Council to Investigate Occupational Dermatitis*

*Reprinted* from *J.A.D.A.*, Vol. 35, November 1, 1947.

As part of its program of therapeutic investigation, the Council on Dental Therapeutics is compiling a list of dentists who are sensitive to procaine (Novocain), Monocaine, Butyn and other drugs used in their practices.

The Council is beginning a study of possible means for the prevention and treatment of occupational dermatitis. The active coöperation of a large number of members of the dental profession will be necessary if the project is to succeed.

The Council asks that all dentists who are subject to occupational dermatitis write to the American Dental Association, 222 East Superior St., Chicago 11, stating the general nature of their ailments. Names will be held in confidence if the writer so requests.